globin were found. The effect of marching and of immersing the hands in ice-water was tried, and with positive results. After immersing the hands for ten minutes the patient began to yawn and shiver and then sweat. He complained of nausea and vertigo; vomited; had pain in the arms. In a few hours the jaundice was more intense. The urine contained albumin and red blood-corpuscles, but no bile-pigment or hæmoglobin. The blood showed no distinct change from the former examinations, so far as the count was concerned, but there were numerous shadows, and staining revealed marked polychromatophilia. The blood-serum showed dissolved hæmoglobin. The patient was seen frequently, and repeated examinations of the urine were made. Bile-pigment was never found, though there were traces of urobilin. The other conditions were unchanged. The author concludes that the patient had a hæmoglobinæmia, usually with abortive paroxysms, the blood coloring matter being altered before it could be excreted in the urine. This might be due to a congenital abnormality of the blood. From the difficulty of investigating such a case during the paroxysms the author raises the question whether in Hayem's cases hæmoglobinæmia may have been overlooked—a thing that is all the more possible because the symptoms of the attack are such as could readily be ascribed to liver disease, especially biliary colic. The treatment of the case was purely symptomatic and tonic, with avoidance of those things that were likely to bring on an attack. Most of the subjective disturbance was due to the enlarged spleen, but with the probability that this was spodogenic, having the object of working over the excess of broken-down blood-corpuscles, extirpation could not be recommended.

Prognosis in Glycosuria and Diabetes.—HIRSCHFELD (*Berliner klinische Wochenschrift*, 1900, No. 26) concludes a thoughtful article on this subject as follows: All authors agree that the prognosis in the milder forms of diabetes is more favorable than has hitherto been considered by the profession.

Relative recovery—*i. e.*, absence of glycosuria with the ingestion of 200 grains of carbohydrates daily—is to be expected when in the beginning of the disease 80 to 85 per cent. of carbohydrates are consumed in the body.

Lessened assimilation of carbohydrates may be observed after influenza, after peculiar colicky attacks (pancreatic colic), and to an especially severe degree in cases with furuncle and gangrene.

Increased severity of glycosuria is sometimes indicated by increased quantity of urine, improvement by diminished excretion.

At times increase of glycosuria coincides with rapid increase of weight, and subsides when the diet is restricted.

The cardiac weakness so common in diabetes is most favorably influenced by mild antidiabetic diet, not wholly excluding carbohydrates.

The Cause of the Presystolic Murmur.—GIBSON (*The Edinburgh Medical Journal*, September, 1900, p. 212) draws attention to the extensive discussion that has taken place in regard to the explanation of the murmurs of mitral obstruction, ever since the discovery of the purring thrill by Corvisart and of the presystolic murmur by Fauvel. He points out that the murmur of mitral stenosis occurs at the apex, and may occur immediately after the second sound—usually termed diastolic; or occurring a little later—therefore sometimes called post-diastolic, or taking place immediately in advance of the first sound—the well-known presystolic murmur. While the existence and character of these murmurs is well recognized, there is considerable difference of opinion as to the explanation of their occurrence.

The usual explanation given for these murmurs is that those of diastolic and postdiastolic rhythm are produced by the active diastole of the left ventricle drawing blood from the auricle, but also aided by the general onward current of the blood, impelled through the pulmonary artery and pulmonary veins by the right ventricle, which in such cases is always hypertrophied. The presystolic murmur has been and is generally considered as due to the contraction of the left auricle, and its absence, or disappearance and reappearance, attributed to varying degrees of weakness of this part of the heart. Gairdner advanced this explanation for the presystolic murmur, and his view was later confirmed and amplified by Tagge and Balfour, and has been adopted by the profession generally. Its acceptance, however, has not been universal.

The first person to express doubt as to the correctness and sufficiency of Gairdner's explanation was Ormerod in 1864. He raised the question whether the presystolic murmur was due to obstruction to the flow of blood from the auricle to the ventricle during the auricular contraction, or to imperfection of the auriculo-ventricular valve, allowing regurgitation during contraction of the ventricle. He expressed doubt whether the contraction of the auricle, slight and momentary as experimentalists then believed it to be, could produce so loud a sound. He saw no objection to the second view, "which assumes that coaptation of all the portions of the valves is not simultaneous; but that when at a certain point of contraction the valve is closed the regurgitant stream is cut off and the cessation of the murmur coincides with the sudden jerk of valvular tension." Barclay, Charlwood Turner, and Dickinson with slight modifications supported the ventricular causation of the presystolic murmur advanced by Ormerod. Brockbank has given his support to the "heretical theory," as he calls it. His view is that "the crescendo murmur of mitral stenosis is produced by blood regurgitating through the stiff, rigid orifice of the narrowed valve, while the orifice which resists closure is being rapidly diminished in area and finally obliterated by the action of a strong force, which on its part increases rapidly in strength with the duration of the bruit."

Brockbank holds that any theory which explains the production of a crescendo murmur must hold good no matter what the condition of the left auricle may be, and he states that the murmur not uncommonly occurs in cases in which the auricle is dilated and weak.

Cowan and Gibbes, in recent articles, with slight variations cast their opinion on the side of the "orthodox theory" of Gairdner. The former holds that the murmur of mitral stenosis is caused in its earlier portion by the blood driven through the thickened valves, by the auricular systole, and in its terminal part by blood flowing into the ventricle while changes in the tension of the valves and alterations in the lumen of the orifice are taking place, the result of the contraction of the ventricle as a whole and of the musculi papillares in particular.

Gibson then proceeds to weigh the arguments advanced in favor of the two theories. He supports Gairdner's theory of the causation of the presystolic murmur. He says that the one simple fact that the presystolic murmur may begin as a diastolic murmur with the second sound, and be continued, without appreciable alteration in its character, up to the first sound, to his mind entirely negatives the regurgitant theory of the presystolic portion, which would require an absolute revolution in our knowledge of the cardiac mechanism.

[With this view I believe a majority of teachers on this side of the water agree.—W. O.]

The Pituitary Gland as a Factor in Acromegaly and Giantism.—WOODS HUTCHINSON has written a series of very interesting articles on the above subject. The early papers were published in 1898, and the more recent ones have just appeared.

Hutchinson points out in his first paper that previous to 1890 the pituitary gland was almost unanimously regarded as a purely vestigial structure and possessing no present value to the organism. In that year interest in its function, if it possessed any, was aroused by the announcement of Souza-Leite, that the gland was markedly enlarged in most cases of the disease known as acromegaly, which was first described by Marie in 1886.

As a result of the analysis of the literature on the subject, and in particular of nineteen cases of acromegaly in which microscopic study of the enlarged pituitary gland was made, Hutchinson draws the following conclusions:

1. That the pituitary body is still functional.
2. That disturbances of its metabolism are the principal factors in both acromegaly and giantism, the difference between the results being simply due to the stage of individual development at which the disturbance of its function begins.
3. That the nature of the overgrowth in both these diseases is primarily on the order of a pure functional hypertrophy; later, however, losing some of the definiteness of its impulse, and either producing immature tissue of a mixed type or resulting in simple hemorrhagic exudation, with either cyst formation or complete breaking down of the tissue mass.
4. That it seems probable, although upon this head the evidence is still uncertain, that some part is played by this body in "dwarfism," rickets, and the dwarf forms of cretinism.
5. That a reflex disturbance of its function may possibly underlie the distrophy accompanying pharyngeal adenoids.
6. That it would appear to be a sort of "growth centre" or proportion regulator of the entire appendicular skeleton.—*New York Medical Journal*, vol. lxvii., 1898, pp. 341, 450, and vol. lxxii., 1900, July 21 and 28.

Tracheal Tugging in Mediastinal Tumor.—AUERBACH (*Deutsche medicinische Wochenschrift*, 1900, No. 8) reports two cases illustrating the necessity of using Oliver's sign in the diagnosis of suspected aortic aneurism. In one case there was a primary cancer of the stomach with metastases in the abdomen and the supraclavicular and bronchial glands. Over the upper part of